

NEW YORK STATE DEPARTMENT OF HEALTH
Bureau of Immunization/Division of Epidemiology

Immunization Requirements for School Attendance Medical Exemption Statement for Children 0-18 Years of Age

NOTE: THIS EXEMPTION FORM APPLIES ONLY TO IMMUNIZATIONS REQUIRED FOR SCHOOL ATTENDANCE

Instructions:

1. Complete information (name, DOB etc.).
2. Indicate which vaccine(s) the medical exemption is referring to.
3. Complete contraindication/precaution information.
4. Complete date exemption ends, if applicable.
5. Complete medical provider information. Retain copy for file. Return original to facility or person requesting form.

1. Patient's Name [REDACTED]
2. Patient's Date of Birth [REDACTED]
3. Patient's Address [REDACTED]
4. Name of Educational Institution Lansing Central School

Guidance for medical exemptions for vaccination can be obtained from the contraindications, indications, and precautions described in the vaccine manufacturers' package insert and by the most recent recommendations of the Advisory Committee on Immunization Practices (ACIP) available in the Centers for Disease Control and Prevention publication, Guide to Vaccine Contraindications and Precautions. This guide can be found at the following website: <http://www.cdc.gov/vaccines/recs/vac-admin/contraindications.htm>.

Please indicate which vaccine(s) the medical exemption is referring to:

- | | |
|--|--|
| <input checked="" type="checkbox"/> Haemophilus Influenzae type b (Hib) | <input checked="" type="checkbox"/> Measles, Mumps, and Rubella (MMR) |
| <input checked="" type="checkbox"/> Polio (IPV or OPV) | <input checked="" type="checkbox"/> Varicella (Chickenpox) |
| <input checked="" type="checkbox"/> Hepatitis B (Hep B) | <input checked="" type="checkbox"/> Pneumococcal Conjugate Vaccine (PCV) |
| <input checked="" type="checkbox"/> Tetanus, Diphtheria, Pertussis (DTaP, DTP, Tdap) | <input checked="" type="checkbox"/> Meningococcal Vaccine (MenACWY) |

Please describe the patient's contraindication(s)/precaution(s) here: [REDACTED] should not receive any of the required vaccines due to her inherited genetic risk, identified by genomic analysis, consistent with presenting clinical picture and family history. Please refer to attached document for additional information

Date exemption ends (if applicable)

A New York State licensed physician must complete this medical exemption statement and provide their information below:

Name (print) Christopher R. Scianna, D.O. NYS Medical License # [REDACTED]
Address White Birch Natural Medicine & Wellness. 51 Ceres St., Portsmouth, NH, 03801

Signature [Signature] D.O. Telephone 315-412-2595
Date 08/11/2019

For Institution Use ONLY: Medical Exemption Status ☐ Accepted ☐ Not Accepted Date: _____

WHITE BIRCH NATURAL MEDICINE & WELLNESS

Christopher R. Scianna, DO
51 Ceres St.
Portsmouth, NH 03801
315-412-2595

I write requesting exemption from New York State vaccination requirements for [REDACTED]
After a thorough evaluation of history and review of his genomic analysis, I conclude this child is at risk for serious physical injury if she is vaccinated in accordance with the NY state vaccination schedule.

[REDACTED] was born with severe unilateral hydronephrosis which corrected itself by two or three years of age. She suffered from febrile seizures during the same time, caused by urinary tract infections. Her family history is significant for: cancer, heart disease, febrile seizures, hearing deficits, food allergies including gluten, multiple environmental and drug allergies and severe adverse reactions to vaccinations causing the death of her father's two-month old brother, as well as a five-month old cousin on his father's side. Her paternal grandmother has also experienced severe vaccine reactions, as well as her father and her paternal aunt who have both been permanently medically exempt from vaccinations since they were five and two-years old, respectively. In addition, the family was awarded compensation from the Vaccine Injury Compensation Program.

From section (l) of NYS guidelines, effective 8/16/19:

"May be detrimental to the child's health means that a physician has determined that a child has a medical contraindication or precaution to a specific immunization consistent with ACIP guidance or other nationally recognized evidence-based standard of care."

Evidence-based guidelines, also called clinical practice guidelines, "are systematically developed statements to assist practitioner and patient decisions about appropriate health care for specific clinical circumstances" (Institute of Medicine; 1990, p. 381; 2001, p. 1512). According to the National Guideline Clearinghouse (NGC), sponsored by the Agency on Healthcare Research and Quality (AHRQ), such "guidelines are not fixed protocols that must be followed" (NGC, 2007) but are intended to identify generally recommended interventions to be considered by a knowledgeable healthcare provider. Guidelines are developed by panels or groups of experienced individuals who carefully weigh syntheses of evidence and the strength of the evidence before developing recommendations for interventions. As each individual is unique and presents different characteristics, professional caregivers must consider the treatment options that are appropriate for the specific situation. Many professional organizations have embarked on developing guidelines for their fields. In rehabilitation, many disciplines may be represented for a specific patient, adding to the quantity of information and options that must be considered.

We have the genetic information necessary to determine the likelihood that these vaccines and their excipients will cause irreversible harm to [REDACTED]

Immunologists concur, autoimmune disorders are not only a result of excessive activation of a normal immune system, but also, activation of a dysfunctional immune system, and according to one of the most prominent figures in vaccine design and development.

Stanley Plotkin, during his sworn deposition in January 2018:

"Vaccines, in effect, are mimicking what happens after natural infections, without causing the complete range of disease that the organism causes."

Ideally our innate and adaptive immune systems work to quickly clear a natural infection and then immediately shut down the immune response, to allow cellular mechanisms to repair collateral damage. This will not be the case for [REDACTED]. Her genetic variation in immune related SNPs, IFH1, IL1B, IL5, IL13, HLA DQA2 and IGF1R, for example, will change the way her immune system reacts, altering the level of response when challenged by a vaccine.

Vaccine adjuvants act as powerful immune-stimulants, designed to activate and sustain a prolonged immune system response, provoking over-activation, resulting in: increased inflammatory cytokines and chemokines, lipid peroxidation, excess glutamate production, and leading to the pathological process of excitotoxicity. The subsequent release of enzymes (phospholipases, endonucleases, proteases) will cause damage to cell structures, the cytoskeleton, cell membrane and DNA. The damage will be slow to repair when considering the epigenetic modifications in her methylation and folate pathways (MTHFD1, MTHFR, and MTHFS, for example).

The potential upregulated immune response in combination with methylation deficits and detoxification issues, leads me to believe [REDACTED] will suffer from excessive microglial activation as these vaccine excipients are likely to invoke a chronic, autoimmune state in her body, causing the microglia to maintain a state of "fight or flight" rather than their neuroprotective, resting state.

Exposure to things like mercury, aluminum, glutaraldehyde and MSG, will act synergistically, amplifying negative effects and perpetuating a state of excitotoxicity. Glutamate levels will rise, increasing permeability of the mitochondria, allowing uncontrolled influx of calcium, leading to swelling and cellular apoptosis.

Administration of these neurotoxic substances will lead to a significant increase in inflammation and must be considered with the compromised metabolic capacity of this child. [REDACTED] will accumulate higher concentrations of these substances and is therefore at significantly increased risk of serious injury as a result of vaccination. Given that the risks outweigh the benefits, I conclude this child should be exempt from any, and all, NYS required vaccinations.

Thank you for your consideration.

Sincerely,



D.O.

Christopher R. Scianna, D.O.

Methylation refers to the process of adding a methyl group (CH₃) to a substrate. Methylation is a central biochemical process responsible for:

- **Removal of homocysteine** (a potentially inflammatory protein)
- **Glutathione formation** (a critical antioxidant)
- **Hormone and neurotransmitter synthesis and breakdown**
- **DNA/RNA synthesis and repair**
- **Nitric oxide formation**
- **Immune cell synthesis**
- **Biotransformation** (breaking down of chemical, heavy metal and xenobiotic toxins)

SNPs include, but are not limited to:

Methylation/Detoxification SNPs:

MTHFR A1298C- C677T, ADA, GP6, BCOMI, TOMM40, SORL1, FOXE1, GGH1, GGH, GSR, GSS, GGT1, GSTM3, HFE, HRH1, HTR2A, MAT1A, MAT2B, MMAB, NQO1, SULT2A1, SLC6A2, SLC6A3, BHMT-02, CAT, MTHFD1L, MTHFD1, F11, NR1I2, FOLR2, FOLR3 NOS2, NOS3, SLC19a1, CETP, DAO, GAD1, MTR, MTRR A66G, MTRR-11, CHAT, FADS1, FADS2, SHMT-1, SHMT-2, SLC25a1, SPPL, PON1, PON2, GSTP1, CYP2D6, SOD2, SOD-3, NAT2, CYP1B1, UGT1A6, UGT1A1, FUT2, CBS C699T, CBS A360A, COMT V158M, COMT H62H, AGT, AGXT, TCN1, DHFR/MSH, MTHFS, ALDH2, CNRI, TKT, TKTL1, PEMT, CHAT, SULT2A1

Neurologic Inflammation/Immune Regulation/Autoimmunity SNPs

HLA-DRB1, IL4, IRF1, LOC105369890, SCN1A, CTLA4, HLA-DQA2, IL1A, IL1B, NLRP3, PLCG2, MTHFR A1298C, NOS2, QDPR, SHMT2, SOD2, SOD3, FGF2, KIT, MAPK1, MAPK14, B9D2, STAT1, STAT3, TFRC, TGFB1, TGFB2, ATG16L1, ATG5, SMURF1, IL4

Associated SNPs that Modulate Response to Vaccination:

<https://www.ncbi.nlm.nih.gov/pubmed/25344690>

TGFβ-1 - Transforming growth factor beta-1 is a growth factor and immune-modulating cytokine.

IL4- interleukin 4, a cytokine that induces differentiation of naive helper T cells (Th0 cells) to Th2 cells. Upon activation by IL4, Th2 cells subsequently produce additional IL4 in a positive feedback loop. It is closely related and has functions similar to IL13.

IL2Ra - Interleukin 2Ra is a receptor for the cytokine IL2 involved in T-regulatory cell differentiation and immunological tolerance. The risk allele is associated with autoimmune diseases, including Type 1 diabetes

IL1 β - Interleukin 1- beta is a potent inflammatory cytokine, which promotes T-cell activation, including that of TH17. The risk allele associated with alopecia, Alzheimer's risk, duodenal ulcers, diabetic nephropathy, joint damage in rheumatoid arthritis, myasthenia gravis, inflammatory bowel disease, periodontitis, ulcerative colitis.

IFI44L - Interferon-inducing gene with antiviral actions.

TRAF 1 - TNF receptor associated 1 – Involved in the activation of the TNF superfamily. Activates NF kappa B.

IRF5 - Interferon Regulatory Factor – Involved in the formation of antiviral cytokine interferon. May be involved in switching macrophages to increase or decrease interferon production. Mutations are seen in IBS, scleroderma.

IL-13 - Interleukin 13 – Anti-inflammatory cytokine derived from TH2 helper cells

TLR4 - gene encodes a protein known as toll-like receptor 4, one member of a family of proteins involved in the innate immune system.

CTLA4 - Cytotoxic T-lymphocyte-associated protein 4 – Transmits inhibitory signal to T-cells. Mutations seen in Grave's disease, Hashimoto's & Type 1 Diabetes.

FOXE1 - Forkhead box E1; thyroid transcription factor – Involved in the formation of congenital hypothyroidism

FCGR2A - Fc Fragment of IgG, Low-Affinity IIa Receptor – Cell surface receptor involved in operational phagocytosis of neutrophils & macrophages.

GF1R - Insulin-like growth factor 1 – Has tyrosine kinase activity. Involved in anti-apoptosis. Mutations may be seen in insulin resistance & type 2 diabetes.

CFH - Complement Factor H – Involved in the restriction of the innate immune defense against microbes.

ATG16L1 - Autophagy related 16 like 1)– Involved in the lysozyme degradation of cells. Mutations are seen in IBS and Crohn's disease. ATG16L1 is involved in the conjugation of phosphatidylethanolamine.

HLA - Histocompatibility complex – Present peptides derived from the endoplasmic reticulum

HLA DQA2 - Major Histocompatibility complex, Class II DQA-alpha – Involved in the CD4 T-cell antigen system via antigen-presenting cell pathways. Peptide loading protein involved in CD4 T-cells.

CHAT - Choline acetyltransferase is a gene that provides instructions for making the neurotransmitter acetylcholine from the B-vitamin choline. Acetylcholine is a primary neurotransmitter of the parasympathetic nervous system, including the vagus nerve.

PEMT - Phosphatidylethanolamine methyltransferase is involved in the conversion of the phospholipid ethanolamine into phosphatidylcholine. Phospholipids are components of cellular membranes, and facilitate vital functions in the brain, liver, intestines and nervous system.

VDR - Vitamin D receptor. VDR is activated by Calcitriol, the active seco-steroid hormone form of Vitamin D. VDR is a nuclear receptor, with numerous post-translational effects, including immunological, calcium-related and hormone-related activations.

FUT2 - Fucosyltransferase 2 — Involved in H antigen formation through oligosaccharide FuC alpha. Associated with intestinal flora imbalance & Crohn's disease. Mutations in FUT2 may predispose towards low concentrations of bifidobacterium. FUT2 may also be involved in Vitamin B-12 levels

PON1 - Paraoxonase 1 is involved in detoxification and influences arylesterase, which catalytically degrades bisphenol A, protects against LDL oxidation and removes lipid peroxides.

GAD1 - Glutamate decarboxylase — Involved in conversion of glutamate to GABA. Associated glutamate excitotoxicity, GABA deficiency

FADS1 and 2 - Fatty acid desaturases are integral in the formation of omega 6 arachidonic acid (AA) and omega 3 eicosapentanoic acid (EPA) and is strongly associated with ceramide and sphingomyelin metabolism, important structural cell membrane lipids. This may be especially useful with chronic inflammation and conditions of autoimmunity

CBS - Cystathione beta synthase — Involved in the conversion of homocysteine into cystathionine via transsulfuration. Involved in glutathione synthesis, capable of preventing oxidative, free radical damage to tissue

FOLR 3 - Folate receptor 3 — Involved in the transport of 5-methylfolate into cells. Mutations are seen in cerebral neuro-degeneration, neural tube defects & RA.

MTHFD1 Methylene tetrahydrofolate dehydrogenase 1 — Involved in the conversion of methylated folate derivatives into purine and thymidylate synthesis, processes important for DNA synthesis and repair.

SHMT serine hydroxy-methyltransferase — Involved in purine synthesis via folate derivatives.

MTHFS methylene tetrahydrofolate cyclo- ligase — Folate derivative involved in the formation of purines, thymidine, and amino acids.

SCN1A - Voltage-dependent sodium channel gene with risk alleles associated with epilepsy and febrile seizures.

SCN2a - Voltage-gated sodium channel gene with risk alleles associated with autism spectrum and seizure disorders. IFI44L - Interferon-inducing gene with antiviral actions

SPP1 - Osteopontin is a gene involved in calcium metabolism and in the formation of calcium-oxalate crystals. Additionally, osteopontin is reportedly a TH1 cytokine.

BCMO1 - Beta carotene monooxygenase is essential in the conversion of β -carotene into Vitamin A. Carriers feature reduced capacity to form Vitamin A from β -carotene. Homozygous carriers of the G risk allele for rs11645428 have shown a 51% decrease in the conversion efficiency of β -carotene into Vitamin A. Homozygous carriers of the A risk allele for rs6420424 have shown a 59% decrease in the conversion efficiency of β -carotene into Vitamin A.

FGA - Fibrinogen alpha chain is involved in the coagulation cascade. The risk allele C is strongly associated with autism spectrum

NR1I2 - Nuclear receptor sub family 2 — Binds to CYP class. Involved in the degradation of xenobiotics.

NAT2 family (N-acetyltransferase) — Involved in the acetylation detox cycles. Acetylation is a phase 2 detoxification reaction. NAT2 mutations reportedly reduce the turnover of acetylation detoxification and may alter the metabolism of certain drugs.

NQO1 - Quinone oxidoreductase - NQO1 detoxifies semi-quinones, which in some forms act as DNA mutagens. NQO1 is essential in the detoxification of various environmental toxins

MTHFR - Methylene tetrahydrofolate reductase — Involved in the formation of 5-methyl folate. The active, methylated folate is central to methylation cycle function, and the “remethylation” of homocysteine. Mutations in MTHFR genes may significantly reduce the rate of homocysteine methylation, leading to an elevation in homocysteine levels, which is a major risk factor for cardiovascular inflammation. Additionally, methylfolate is an essential substrate for neurotransmitter synthesis as well as DNA synthesis and repair.

MTRR - 5-methylenetetrahydrofolate homocysteine methyltransferase reductase — Involved in the recycling of homocysteine into methionine. MTRR methylates vitamin B-12, which is the major cofactor for this junction in the methylation cycle. MTRR works in concert with the MTHFR gene. SLC19A1 is the folate transporter.

TCN1/TCN2 - Transcobalamin II — Involved in the transport of cobalamin into cells. May indicate B-12 deficiency.

MAT1A - Methionine adenosyl transferase is involved in the conversion of the amino acid methionine into S-adenosyl methionine, SAME, which is the primary methyl donor. 70% of one-carbon methylation reactions involve SAME as the cofactor, including reactions with most methyltransferase

DAO - Di-amino oxidase — Enzyme involved in the degradation of histamine. Involved in dopamine synthesis. Regulates L-Serine in the brain. Mutations are seen in schizophrenia & bi-polar disorders. Histamine is an immune compound as well as a neurotransmitter.

KNG1 - Kininogen — Involved in blood clotting. GP6 (Glycoprotein 6) — Involved in collagen-induced platelet aggregation

ALDH2 - Aldehyde dehydrogenase — Involved in the detoxification of alcohol, specifically in the oxidation of aldehydes into carboxylic acids. Deficiency of ALDH2 enzyme activity is believed to increase levels of acetaldehyde and contribute to its toxicity.

SOD2- superoxide dismutase — Mitochondrial antioxidant of the manganese class, involved in detoxification.

SOD3 - Superoxide dismutase-3 is an antioxidant that quenches the superoxide anion

DHFR - Dihydrofolate reductase - Folate derivative involved in purine synthesis

FCER1A -Fragment of IgE High Affinity — Involved in allergic histamine response.

Excipient Related SNPs for [REDACTED]

2-Phenoxyethanol

DUSP1 G*892A, ERCC2/XPD G16331T, IL1B C-511T, IL8 A4383C, PTGS1/COX-1 A-287G, XRCC1 A194T, XRCC1 G399A

A-Tocopheryl Hydrogen Succinate

ABI2 C204272090T, APP .T27272159C, ERCC1 G540K, ERCC4 S835S

Acetone

IGLL1/14.1 T4513C, NR3C1 C3843271A

Alcohol

DUSP1 G*892A, ERCC2/XPD G16331T, IL1B C-511T, IL8 A4383C, PTGS1/COX-1 A-287G, XRCC1 A194T, XRCC1 G399A

Aluminum

IL4 C-33T, TCN1 G4939288A

Aluminum Hydroxide

IL4 C-33T, TCN1 G4939288A

Aluminum Hydroxyphosphate Sulfate

FBP1 T97390288C, IL4 C-33T, TCN1 G4939288A

Aluminum Phosphate

FBP1 T97390288C, IL4 C-33T, TCN1 G4939288A

Aluminum Potassium Sulfate

IL4 C-33T, TCN1 G4939288A

Aluminum Salts

IL4 C-33T

Aluminum Sulfate

IL4 C-33T, TCN1 G4939288A

Amino Acids

BAG1 A13581C, GAD1 (GAD) A48604A, GAD1 (GAD) C10180T, GAD1 (GAD) C14541T, GAD1 (GAD) C18360T, GAD2 (GAD) G26474809T, SAA1 A18285774T, SHMT1 A23836G, UGT1A1 G179250T

Aminoglycoside Antibiotic

TH T1090C

Amphotericin B

BDNF C27677041T, SLC6A4 A28525011C

Anhydrous Lactose

B4GALT1 A47485G, B4GALT1 C33170362T, LCT A1639S

Ascorbic Acid

HFE 6382T>G, HFE 8828T>C, HFE H63D

Benzethonium Chloride

CHRFAM7A/CHRNA7 C78732T, CHRFAM7A/CHRNA7 G121573A, CHRND A233400074G, CTSH A6975G, CTSL1 A8782406G

Bovine Serum Albumin

SAA1 A18285774T, UGT1A9 Asp256Asn

Calcium Chloride

THBS2 A169617726C

Castor Oil

CNR2 T24221834G, IL4 C-33T, PTGER3 G90650T, VCAM1 A71159137G

Chicken Protein

UGT1A1 G179250T

D-Mannose

MBL2 G54A, VDR TAQ, VDR VDR:BsmI

Dextrose

IL7 T23148959C, LEP G127881349A, LGALS1/GAL1 A17468287G, MLXIPL or MLX G24H, SREBF1/SREBP C10586T, SREBF1/SREBP G11566A

Dipotassium Phosphate

FBP1 T97390288C, KCNA3 T*232G

Disodium Phosphate

FBP1 T97390288C

Dulbecco's Modified Eagle Medium

KCNA3 T*232G, PTCH1 P1164L, SERPINE1 T16067G

FD&C Yellow #6 Aluminum Lake Dye

CD19 Pro235Pro

Formaldehyde

ALDH2 T35023C, IL10 T206946897C, IL2 T4671G

Gelatin

BAG1 A13581C

Gentamicin Sulfate

NFE2 A54687232C, NFE2 G16855614A

Glutaraldehyde

ALDH2 T35023C

Glyphosate

ABP1 P574P, AGT 11216C>T, AGT 13828T>C, AGT 17006C>A, AGT 5465G>A, AGT 5538T>C, AGT 5978A>G, AGT M235T/C4072T, AGXT I340M, AGXT P11R, AGXT2 A35035579G, AGXT2 A35045745G, AGXT2 C35044298G, AGXT2 V140I, AGXT2 V498L, BCKDHA C5472T, BCKDHB C229524T, DAO G8864A, DAO T887G, DAO T9891G, DBT G384S, DDO C110717493T, DLD T19214C, GLDC A55715G, GLDC C118216T, HAO1 A7873112G, HAO1 C7897049T, HAO1 T7905947C, HOGA1 T20638C, PDHX V271V

Human Serum Albumin

SAA1 A18285774T, UGT1A1 G179250T

Hydrocortisone

CD40 T44746982C, CYP2B6 C26570T, CYP2B6 G29435A, CYP2B6 L262A, CYP2B6 Q172H, CYP2B6 T1421C, CYP2B6 T23499C, IL4 C-33T

Kanamycin

NFE2 A54687232C, NFE2 G16855614A

L-Cystine

CTH A11886G, CTH G25229T, CTH T8763C

L-Glutamine

GAD1 (GAD) A48604A, GAD1 (GAD) C10180T, GAD1 (GAD) C14541T, GAD1 (GAD) C18360T, GAD2 (GAD) G26474809T

L-Tyrosine

TH T1090C, TH T7517C, TYR G105007A

Lactose

B4GALT1 A47485G, B4GALT1 C33170362T, LCT A1639S

Magnesium Stearate

IFNGR1 -611G>A, SREBF1/SREBP C10586T, SREBF1/SREBP G11566A

Magnesium Sulfate

CACNA1C A2729632G, CACNA1C T709021C

Mercury (Thiomersal/Thimerosal)

COMT V158M, GCLM A7135G, GGT1 T17549C, IL4 C-33T, IL6 T22768707G, MTHFR A1298C, PON1 Q192R, SEPP1 42800706C>T, TF 24053G>A, TLR1 S602I

Monopotassium Glutamate

KCNA3 T*232G

Monopotassium Phosphate

FBP1 T97390288C, KCNA3 T*232G

Monosodium Glutamate

GAD1 (GAD) A48604A, GAD1 (GAD) C10180T, GAD1 (GAD) C14541T, GAD1 (GAD) C18360T, GAD2 (GAD) G26474809T

Monosodium Phosphate

FBP1 T97390288C, PTCH1 P1164L, SERPINE1 T16067G

Neomycin (Neomycin Sulfate)

NFE2 A54687232C, NFE2 G16855614A

Non-Viral Proteins

CAT A12175G

Nonylphenol Ethoxylate

CYP1A2 1545T>C, CYP1A2*1F C164A, ESR1 T152119119G

Octylphenol Ethoxylate (Triton X-100)

CDH5 C66425176T, NF1 T29495895G, P2RX4 S42G

Ovalbumin

UGT1A1 G179250T

Phenol USP

PAH C45031T, PAH C81837T, PAH G37636A, PAH G735A, PAH T32409C

Phosphate-Buffered Saline

FBP1 T97390288C, PTCH1 P1164L, SERPINE1 T16067G

Polacrillin Potassium

GSR A43851G, KCNA3 T*232G

Polysorbate 20

EGFR D994D, PTGS1/COX-1 A-287G, PTGS2 A186649221G

Polysorbate 80

CYP3A4*1B A392G, ICAM1 L469G

Potassium Chloride

KCNA3 T*232G

Protamine Sulphate

HRH1 T*1687C, HRH1 T-17C

Purified Capsular Polysaccharide

IRF3 G50168871A

Sodium Borate

SLC4A11 R161R

Sodium Chloride

PTCH1 P1164L, SERPINE1 C38812087T

Sodium Deoxycholate

EGFR D994D, RAF1 G12626516A, STX1A T73133241G,

Sodium Pyruvate

HFE 6382T>G, HFE 8828T>C, HFE H63D, SOD2 406+816G>T, SOD2 V16A

Sodium Taurodeoxycholate

EGF A110834110G

Sorbitol

DUSP1 G*892A, ERCC2/XPD G16331T, IL1B C-511T, IL8 A4383C, PTGS1/COX-1 A-287G, SORD A45317915C, XRCC1 A194T, XRCC1 G399A

Urea

EGF A110834110G, EGF M708I

Yeast Protein

GLS2 G56865056C, GLS2 L581P

ADDITIONAL SUPPORTING DOCUMENTS:



To whom it may concern,

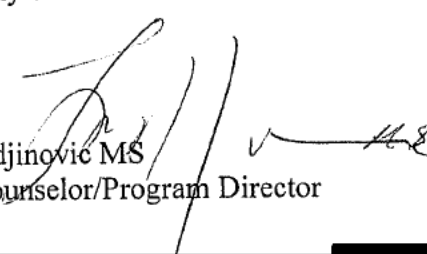
Re: Family members of [REDACTED]

This letter has been prepared for the first-degree and second-degree relatives of [REDACTED]. [REDACTED] death has been confirmed to be a vaccine-related death (Fed. Cl. 1989). [REDACTED] siblings each experienced allergic reaction following routine vaccinations and have been advised to discuss personal and family vaccine reactions with their physicians.

Adverse drug reactions occur at standard doses used in the treatment are well documented. The clinical application of pharmacogenomics in the study of drug related responses to genetic variants in HLA and cytochrome 450 genes offer preliminary evidence of risk associations. Unfortunately, the role of familial genetic variants in vaccine-related deaths have not be evaluated. Severe reactions leading to death must be carefully considered and not set aside when genomic risk data is limited. Additional genomic research is needed to offer family members of [REDACTED] guidance regarding possible severe adverse risk to vaccines.

In s [REDACTED] confirmed vaccine-related death supports caution in following routine vaccine recommendations for first-degree and second-degree relatives until pharmacogenomic testing becomes available to guide family members. Immunization could well be extremely determinantal to their health.

Sincerely,

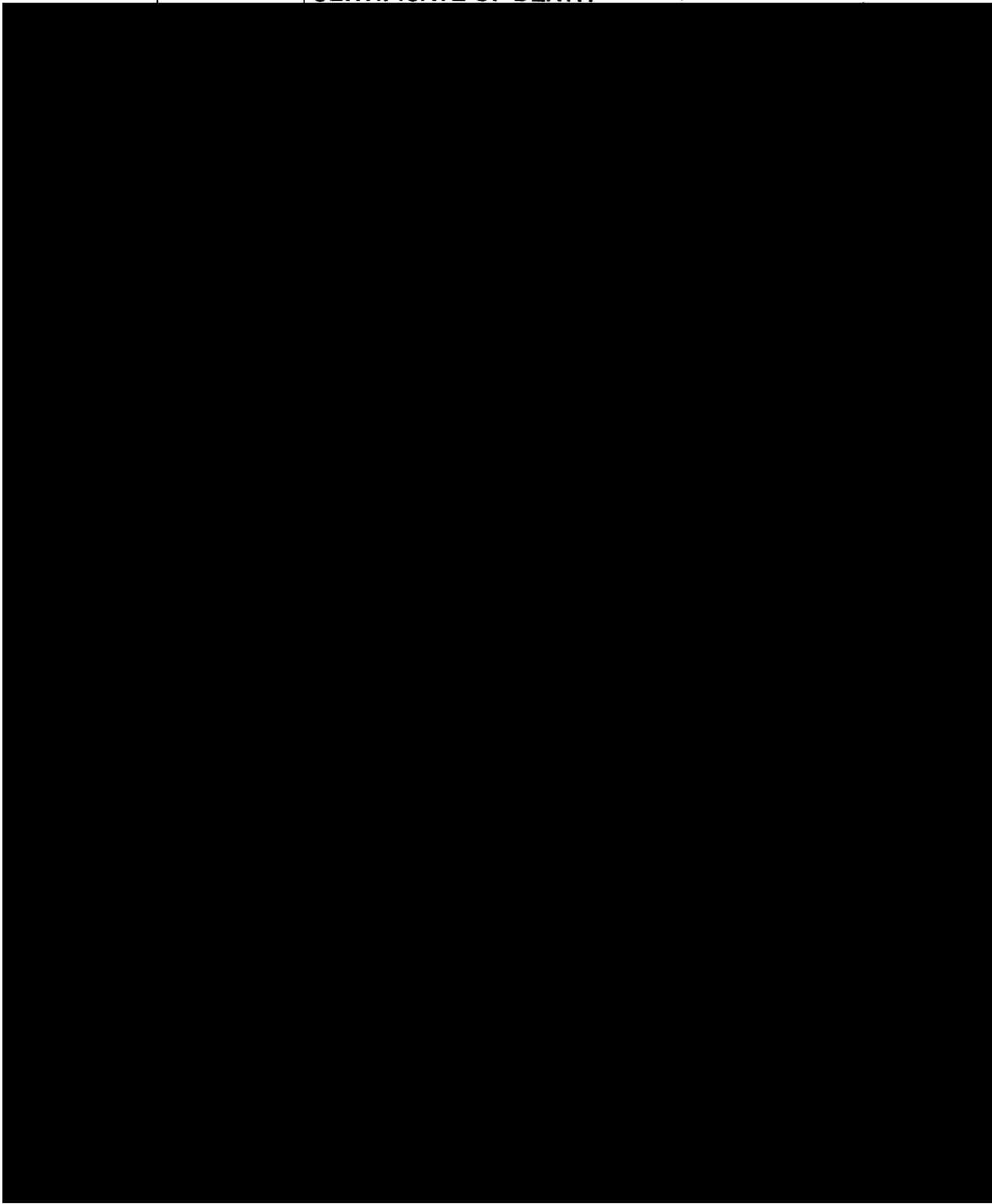

Luba Djurdjinovic MS
Genetic Counselor/Program Director

1. [REDACTED]
2. Osawa M et al. Sudden infant death after vaccination: Survey of Forensic Autopsy Files. American Journal of Forensic Medical Pathology 2019 Sept 40 (3) 232-237/

CENSUS TRACT			SUB-DIVISION	

RECORDED DISTRICT
363
REGISTER NUMBER
3

NEW YORK STATE
DEPARTMENT OF HEALTH
CERTIFICATE OF DEATH





United Health Services Hospitals

July 23, 1997

To whom it may concern:

[REDACTED] has been denied the administration of vaccines because of a unique and extraordinary family medical history.

His bother died at age two months following a serious reaction to DPT and OPV, and [REDACTED] himself had severe side-effects following childhood vaccines. Immunizations could well be extremely detrimental to his health.

Please feel free to contact me at 762-2468 if there are any questions regarding this serious matter.

Sincerely yours,

Cheryl B. Kerr, MD

